

**VALIDATION OF KREB'S SCORING SYSTEM
FOR INTRAPARTUM CST IN DETECTING
COMPROMISED FETUS COMPARED TO
SCALP BLOOD pH ESTIMATION**

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**MADRAS MEDICAL COLLEGE
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CERTIFICATE

This is to certify that this dissertation entitled “**VALIDATION OF KREB’S SCORING SYSTEM FOR INTRAPARTUM CST IN DETECTING COMPROMISED FETUS COMPARED TO SCALP BLOOD pH ESTIMATION**” is a bonafide work done by **Dr.R.JEYA NIRMALA** post graduate student in **M.D. (OBSTETRICS AND GYNAECOLOGY)** under my over all supervision and guidance at The Institute of Social Obstetrics and Government Kasturba Gandhi Hospital, Madras Medical College, Chennai in partial fulfillment of the regulations of Tamilnadu Dr.M.G.R. Medical University for the award of M.D. Degree in Obstetrics and Gynaecology.

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INTRODUCTION

The practice of modern obstetrics involves rendering of optimal care to the mother and the fetus. The birth process has been witnessed and recorded for millennia, and the care rendered to the laboring patient has developed over that length of time.

Significant intrapartum fetal asphyxia occurs in approximately 20 per 1000 live births. The ultimate goal of intrapartum assessment of fetus is to identify accurately the fetal problems in labour, which if uncorrected could lead to death, or short or long term morbidity so that intervention may be made to alleviate the condition.

Fetal heart rate monitoring was started in 1950s. By 1980, fetal heart rate monitoring has become standard practice during labour. Edward Hon was the first to recognize that the fetal heart rate pattern could predict the neonatal outcome as determined by 5 min. APGAR score.

Sahling pioneered the technique of fetal blood sampling in 1960 in the intrapartum fetal assessment. The fetus in labour is not a passive onlooker with no threat posed to its existence (Buckall and Wood .1985) and our understanding of the risk of fetus has only recently been elucidated in terms of biochemical insult and fetal response (Kjellmer, 1988).

The original concept of Little (1862) and Haldane (1922) that cerebral palsy and fetal death are caused by hypoxia at the time of delivery, have been challenged by the epidemiological data showing that by the time labour begins, the majority of cerebral damage has already taken place (Nelson and Ellenberg, 1996) and even severe perinatal asphyxia does not result in cerebral palsy.

There is good evidence that the metabolic acidosis that develop during labour can lead to cerebral damage and fetal death (Kjellmer, 1988) and that such insults are not limited to the pregnancies, determined as high risk by virtue of maternal diseases or antenatal placental compromises (Skyes et al, 1983).

Fetal asphyxia is a condition of impaired blood exchange leading, if it persists to progressive hypoxaemia and hypercapnia with metabolic acidosis. Various methods have been employed for intrapartum fetal surveillance. Of these Krebs intrapartum scoring system has been used as a method to find out fetal hypoxia and has been compared to scalp pH examination to detect fetal hypoxia so that intervention can be made at the right time to prevent this complication.

METHODS OF INTRAPARTUM FETAL MONITORING

1. **INTERMITTENT FETAL AUSCULTATION:** It is done using standard stethoscope or Pinnard's fetoscope or Doppler USG device. Every Kennedy of Rotunda Hospital introduced the fetal heart auscultation.

Fetal heart rate should be counted for 1 min., every 15 to 30 min., immediately after the contraction. It does not allow for accurate assessment of variability or periodic changes, when the risk factors for uteroplacental insufficiency are present during the first stage of labour. Intermittent auscultation must be done every 15 min. in first stage of labour and every 5 min. in second stage, when there is a risk factor. When there is no risk factor it should be done every 30 min. in first stage and every 15 min. in second stage of labour.

2. **ELECTRONIC FETAL HEART RATE MONITORING:** It can be done in 2 ways: Internal method using scalp electrode, external method using ultrasound transducer. Internal method of fetal heart rate monitoring requires direct contact of the electrode with the fetus and hence can be performed after the membrane ruptures.
3. **FETAL SCALP BLOOD SAMPLING:** In all cases of suspicious fetal heart rate tracing in labour, fetal blood sampling (FBS) is required.

4. **FETAL SCALP STIMULATION TEST:** Fetal scalp is stimulated by pinching with a tissue forceps, while the CST is observed for an acceleration (Clerk et al, 1984). If an acceleration is observed it is highly predictive of non-acidotic fetus.
5. **FETAL ACOUSTIC STIMULATION TEST (FAST):** Sound stimulation with an artificial electronic larynx results in acceleration of fetal heart rate of non-acidotic fetus (Smith et al ,1986). About 50% of the fetus fail to respond with an acceleration will have a scalp pH <7.20. These stimulation tests will be useful in identifying the non-acidotic fetus. But further evaluation of non responders by FBS would be necessary.
6. **PRESENCE OF MECONIUM IN LABOUR:** The significance of presence of Meconium in labour is controversial. The risk of thick Meconium particularly is in association with post term pregnancy and IUGR, and has been associated with the risk of fetal acidemia. Despite the lack of a clear relationship between the Meconium staining of liquor and fetal acidosis in the absence of fetal heart rate changes, we can not conclude that the presence of Meconium is not a threat to fetus even if fetal heart is normal. If the fetal heart rate pattern is abnormal the presence of Meconium is associated with higher chance of baby being acidotic and needing resuscitation at birth (Steer et al, 1989)

7. FETAL ELECTRO CARDIOGRAPHY: Internal monitoring of fetal heart rate shows changes in ST segment and PR interval of ECG, as an adjuvant to conventional intrapartum fetal monitoring.
8. FETAL PULSE OXIMETRY: Assessment of fetal oxyhaemoglobin saturation by a unique sensor after the membranes have ruptured is another method of fetal surveillance. Saturation values of below 30% when persistent for 2 min. were associated with the increased risk of potential fetal compromise.
9. INTRAPARTUM DOPPLER VELOCIMETRY: Doppler analysis of the umbilical artery has been studied as another potential adjuvant to fetal monitoring. Abnormal Doppler wave forms may signify pathological umbilical placental vessel resistance. However Farell and coauthors concluded that this technique was a poor predictor of adverse perinatal outcome.

PATHOPHYSIOLOGY OF FETAL HYPOXIA

Oxygen supply to the fetus depends principally on the adequacy of uterine perfusion, placental gas transfer and fetal circulation.

Even though oxygen tension in the umbilical venous blood is low, fetal tissue oxygenation is more than adequate because of

1. Hb concentration in the fetus is higher than in adults.
2. At the tissue level fetal Hb delivers more oxygen.

These physiological changes make the fetus resistant to mild and moderate hypoxia. However when the decrease in umbilical venous oxygen tension becomes severe, fetal hypoxia occurs. The fetus switch on to anaerobic metabolism, leading to the accumulation of lactate and a consequent fall in tissue and blood pH.

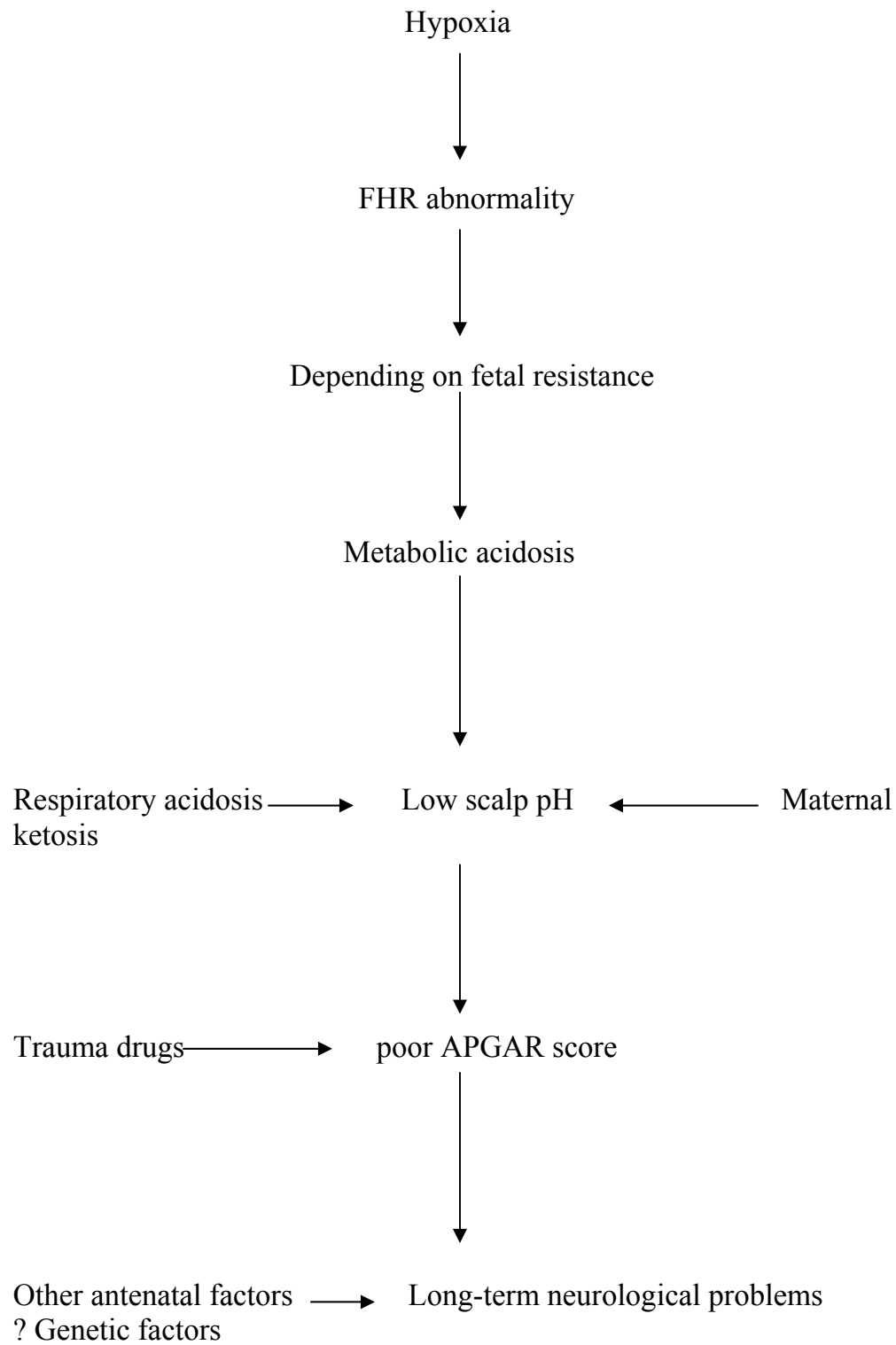
The fetal response to hypoxia depends on the acuteness of onset and its severity and duration. The initial fetal response to gradually developing hypoxia is an attempt to increase and redistribute cardiac output to vital organs like brain and heart. Hence there is an increase in fetal heart rate. This may be followed by a decrease or low of variability and an absence of accelerations due to hypoxia of the brainstem centre; with worsening oxygen supply, hypoxic depression of the myocardium occurs.

The fetal response in an individual case is modified by the compensating mechanisms, which depend upon the reserve capacity of the fetoplacental unit. However, when the hypoxia is acute, the initial reflex response is a decrease in FHR, manifesting as prolonged bradycardia or recurrent decelerations, caused initially by chemoreceptor mediated vagal stimulation and subsequently by hypoxic myocardial depression. These changes in FHR form the basis of fetal heart monitoring as the most common method of intrapartum welfare of the fetus.

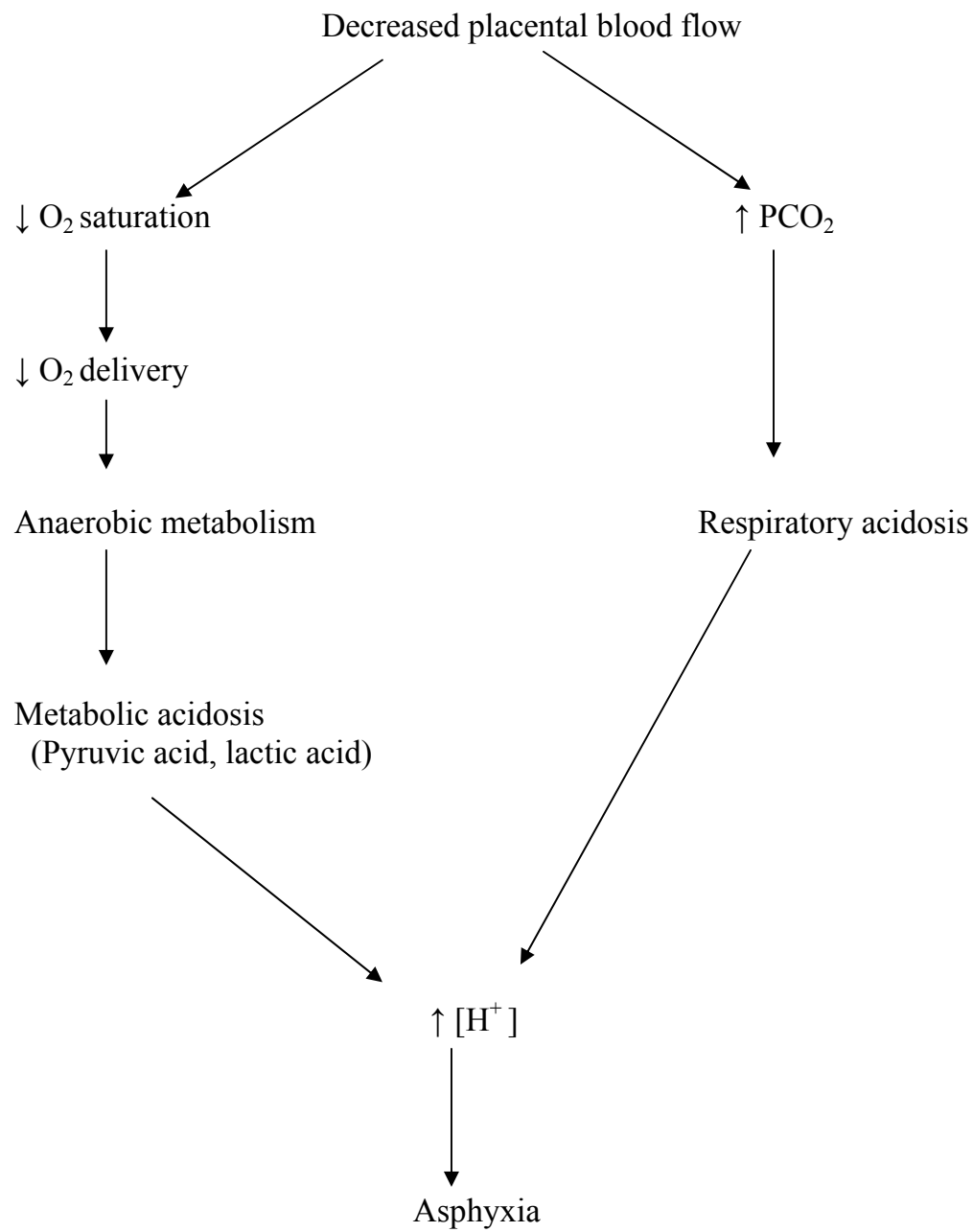
With poor placental transfer of gases, Carbon-di -oxide accumulates in fetus, resulting in respiratory acidosis. The base deficit remains normal or only slightly increased. This form of hypoxia is easily reversed if Carbon-di -oxide is eliminated (Ingemarson and Arulkumaran, 1986).

However, further prolongation of fetal hypoxia leads to anaerobic glycolysis and metabolic acidosis. Metabolic acidosis cannot be reversed until oxygen delivery to fetal tissues is reestablished. Hence when FHR monitoring suggests fetal hypoxia, biochemical monitoring of the fetal acid base status improves the diagnostic accuracy.

RELATIONSHIP BETWEEN HYPOXIA, INTRAPARTUM FETAL MONITORING AND NEONATAL OUTCOMES



PATHOPHYSIOLOGY OF METABOLIC AND RESPIRATORY ACIDOSIS



REGULATION OF FETAL HEART RATE

1. Autonomic nervous system :

Both sympathetic and parasympathetic systems act in coordination to control FHR. Stimulation of sympathetic system increases the FHR, while parasympathetic system stimulation produces a vagally mediated fall in FHR.

2. Baroreceptors and chemoreceptors :

They play a role in the regulation of FHR. They produce different types of decelerations. A reduction in blood pressure will increase the FHR by inhibiting the vagal activity. An increase in BP induces a reduction in FHR mediated by increased vagal tone.

Chemoreceptors respond to changes in the partial pressure of dissolved oxygen in fetal blood.

3. Other influences :

Hormonal factors include the fetal adrenal glands which secrete adrenalin and noradrenaline with ionotropic and chronotropic effects on heart.

REVIEW OF LITERATURE

Marsac , a French obstetrician was probably the first to observe fetal heart sounds on December 22 ,1822. Jean Alexander Lejumeau, Vicomte de Kergaradu read his monograph “memoir sur auscultation appliqué a l’étude de la grossesse” in Royal academy of medicine in Paris.

Laennec, a physician working in Paris in 1806, was the father of the technique of auscultation of adult heart and lungs. Francois mayor (1818) reported the fetal heart audibility different from the maternal pulse. Kennedy (1843) reported the change in fetal heart rate during pregnancy and labour. Auer Friedrich was the first to design fetal stethoscope in 1834. Bepaul modified this. Pinnard's version appeared in 1876. Winkel in 1893 empirically set the limits of normal heart rate 120-160 bpm.

In 1833, Kennedy in Dublin published his monograph on obstetrical auscultation .In 1953 Gunn and Woods reported the amplification and recording of fetal heart sounds in the Proceedings of Royal society of Medicine. In 1958, Hon pioneered electronic fetal monitoring in USA. Hohl (1833) and Hunter (1862) believed that tachycardia was associated with maternal fever and fetal compromise.

Kennedy (1833) thought that most ominous fetal heart signs were slowness of its return following a contraction. Beginning in 1958 an electronic system, the current fetal heart rate monitor, was developed by Hon

and others to detect and plot heart rate automatically in a continuous fashion. The fetal heart rate tracing could then be correlated with continuously recorded uterine activity data.

In 1903, Von Winckel suggested that a fetal heart rate above 160 or below 100 bpm should be regarded as evidence of distress.

Hammacher (1962) developed the first antenatal Cardiotocograph equipment with the phonocardiograph and also reported on the fetal heart rate characteristic associated with fetal compromise in abnormal pregnancies. Freeman and Lee (1975) introduced Non-stress test to describe fetal heart rate acceleration in response to fetal movements as a sign of fetal health.

The largest well randomized trial, performed at Dublin's maternity hospital between 1981 and 1983, compared continuous internal monitoring with auscultation every 15 minutes in the first stage of labour and after each contraction in the second stage. No differences were found in the intrapartum or neonatal death rates or in low Apgar score. A twofold higher rate of neonatal seizures and abnormal neurological examination were observed in the auscultation group. Hon (1967) formulated a predictive scoring system based on fetal heart rate pattern noted prior to 20 minutes of delivery. The predictive value of a normal fetal heart rate pattern in detecting a neonate with APGAR greater than seven was 99 percent. The abnormal fetal heart rate pattern predicted a depressed neonate with 67 percent accuracy.

The admission test has a high predictive value for fetal wellbeing (98.7) percent and high specificity (99.4%) but rather a low predictive value of an abnormal test (40%) and low specificity (23.5%). The admission test cannot be expected to predict fetal distress that develops several hours later if fetal conditions were satisfactory on admission (Ingemarsson, 1993). If the admission test is normal and Oxytocin and Epidural analgesia have not been used, the risk of fetal hypoxia occurring in the next three hours is low. When it occurs, such hypoxia is likely to be from acute events. Example placental abruption or cord prolapse (Ingemarsson and Arul kumaran, 1989).

In 1961, Saling introduced scalp blood pH estimation for the direct assessment of fetal well being during labour. Tejani and associates (1975) correlated fetal heart rate tracings scalp pH values. The scalp blood pH greater than 7.25 was associated with fetal heart rate tracings of normal acceleration and early deceleration in 93 percent of cases. Kuble and colleagues (1969) studied the association of fetal heart rate pattern with fetal scalp blood pH. In patients with fetal heart pattern during the 20 minutes preceding the blood sampling , the frequency of pH values was greater than 7.25 was 94% when fetal heart rate acceleration was present.

Krebs et al, 1979, found that the fetal heart rate raising scores of 8 -10 were associated with normal fetal scalp pH values of greater than 7.25.

6 -7 Score indicates pH 7.2 – 7.25

5 and less than 5 indicate pH less than 7.2

Clark et al investigated the predictive value of fetal heart rate accelerations evoked by stimulation at the collection of fetal scalp blood samples. Hutch et al studied the relationship between the fetal heart rate pattern and fetal transcutaneous PO₂ measurement. Blood lactate levels may be used in fetus and newborn as an indicator of acidosis in labour and delivery (Smith et al 1983, Sudan et al 1984). In predicting neurological damage, the assessment of scalp blood lactate level is more important than scalp pH (Myer et al 1981).

The fetal ST (waveform) Analyzer (STAN) has been proven of value in clinical trials when used in conjunction with CST interpretation (Amer-Wahlin et al 2001) with reduction of operative delivery rates for fetal distress and the incidence of metabolic acidosis.

Fenton and Steer ascribed the recognition of Meconium passage during labour as an indicator of fetal distress to Schwartz in 1858. They found that the combination of thick Meconium and fetal heart rate under 100 beats per minute was associated with the perinatal mortality of 22.2%. They also noted that Meconium alone, or the slowing of the heart rate alone, was not a sufficient indicator of fetal distress. Walker in (1954) found that if thick Meconium is associated with abnormal fetal heart pattern, there is a higher risk of acidosis (Miller et al, 1995).

FETAL HEART RATE MONITORING USING CARDIOTOCOGRAM (CST)

A cardiotocogram is used to produce a continuous recording of FHR and uterine contractions known as cardiotocograph.

Electronic fetal monitors measure the fetal heart rate and uterine contractions by means of external transducers using the Doppler ultrasound and a strain gauge.

Good recording of FHR is dependent on correct placement of the transducer on the abdomen. This is not always achieved and constant readjustment is often achieved as the fetus moves inn utero.

LIMITATIONS

The tocodynamometer gives only a relative indication of contraction strength and the recording is attenuated if the mother is obese or the transducer is poorly applied.

The following are the interpretation of fetal heart rate recording:

1) Baseline heart rate

This is derived from a line passing through a saw toothed fluctuations of the trace. Normal baseline rate at term is between 110 and 150 beats /minute. (FIGO guidelines1987).

2) Baseline variability

Normal variability is between 10 and 25 beats/minute. It is measured by the difference between the two lines drawn through the highest and lowest points of the trace in any one minute segment.

3) Acceleration:

Transient increase in fetal heart rate associated with uterine contractions or fetal movements are known as accelerations and usually indicates that the fetus is adequately oxygenated i.e. rise in fetal heart rate greater than 15 bpm lasting for 15 seconds or more.

4) Deceleration:

Fall in the fetal heart rate greater than 15 beats per minute from the baseline lasting for 15 seconds or more.

5) Early deceleration:

Deceleration, where the lowest point of fetal heart rate occurs within 20 seconds of peak of contractions.

6) Late deceleration:

Deceleration, where the lowest point of fetal heart rate occurs more than 20 seconds after the peak of contractions.

7) Variable deceleration:

Variable deceleration may begin before the onset of contraction, with onset of contraction, following the onset of contraction. They are described as mild, moderate and severe. They are described as severe when the drop is greater than 60 beats per minute and lasts greater than 60 seconds and as 'mild' when the drop lasts less than 60 seconds.

8) Tachycardia:

The sustained rise in heart rate greater than 160 beats per minute.

9) Bradycardia:

The sustained fall in heart rate less than 100 beats per minute.

CLASSIFICATION OF INTRAPARTUM CST

1. NORMAL REASSURING :

- Baseline heart rate 110 to 150 beats per minute.
- Baseline variability 5 to 25 beats per minute.
- Presence of acceleration of at least two each lasting for greater than 15 seconds and greater than 15 beats per minute
- Absence of deceleration

2. SUSPICIOUS / EQUIVOCAL :

- Absence of accelerations for greater than 40 minutes.
- Baseline heart rate 150 to 170 beats per minute or 100 to 110 beats per minute.
- Absent baseline variability (< 5) for greater than 40 minutes, with normal baseline and no deceleration.
- Variable decelerations less than 60 beats per minute for less than 60 seconds
- Transient prolonged Bradycardia less than 80 beats per minute for greater than two minutes.

3. PATHOLOGICAL / OMNIOUS :

- Baseline fetal heart rate greater than 150 beats per minute with absent variability and / or repetitive late or variable decelerations.
- Absent baseline variability (<5) for more than 90 minutes.
- Repetitive late decelerations.
- Prolonged Bradycardia (less than 80 beats per minute for more than 10 minutes).
- Complicated variable deceleration (>60 beats per minute lasting more than 60 seconds).
- Sinusoidal pattern with no acceleration.

ABNORMAL FETAL HEART RATE PATTERNS

1. Tachycardia :

Mild - fetal heart rate between 160 and 180 beats per minute.

Severe - > 180 beats per minute

Causes:

- Physiological i.e. preterm
- Pathological
- Anemia
- Fetal hypoxia
- Maternal fever
- Atropine
- Maternal hypotension

The key feature to distinguish fetal compromise and association with tachycardia is concomitant fetal heart rate deceleration.

2. Bradycardia

Mild - fetal heart rate between 100 and 119 beats per minute.

Moderate - fetal heart rate between 80 and 100 beats per minute.

Severe - fetal heart rate < 80 beats per minute.

Causes:

- Head compression in second stage of labour
- Cord prolapse
- Uterine hyperstimulation
- Abruptio placenta
- Maternal hypoperfusion

3. Sinusoidal pattern:

It indicates severe fetal hypoxia. The features are:

- Regular oscillations above and below a normal baseline rate (120 -160 beats per min.)
- Amplitude of 5 – 15 beats per min.
- Fixed or flat short term variability

- Long term variability frequency of 2 – 5 cycles per min.
 - Absence of acceleration
4. Decreased baseline variability :

Reduced baseline heart rate variability is the single most reliable sign of fetal compromise especially if associated with decelerations.

- Normal variability: 10 – 25 beats per min.
- Absent variability: less than 5 beats per min.
- Reduced variability: 5 – 10 beats per min.
- Increased or salutatory pattern: >25 beats per min.

Causes:

- a) hypoxia
- b) sleep pH of fetus
- c) prematurity
- d) drugs – magnesium sulphate
- e) cardiac arrhythmias

5. Early deceleration :

They were described Hon (1958). Early decelerations are seen in head compression and the mechanism is one of reflex slowing mediated by the Vagus nerve with release of Acetylcholine at the Sinoatrial node commensurate with the pressure applied to the fetal vertex. These early decelerative changes are innocuous and can be observed throughout labour without alteration of the fetal condition.

6. Late deceleration :

It indicates uteroplacental insufficiency and decrease intervillous exchange between mother and fetus with intermittent fetal hypoxia. This was the first fetal heart rate consequence of uteroplacental induced hypoxia (Murata, 1982).

During the course of progressive hypoxia as acidosis develops, baseline fetal heart rate variability disappears.

Mechanism: a) hypoxic myocardial depression

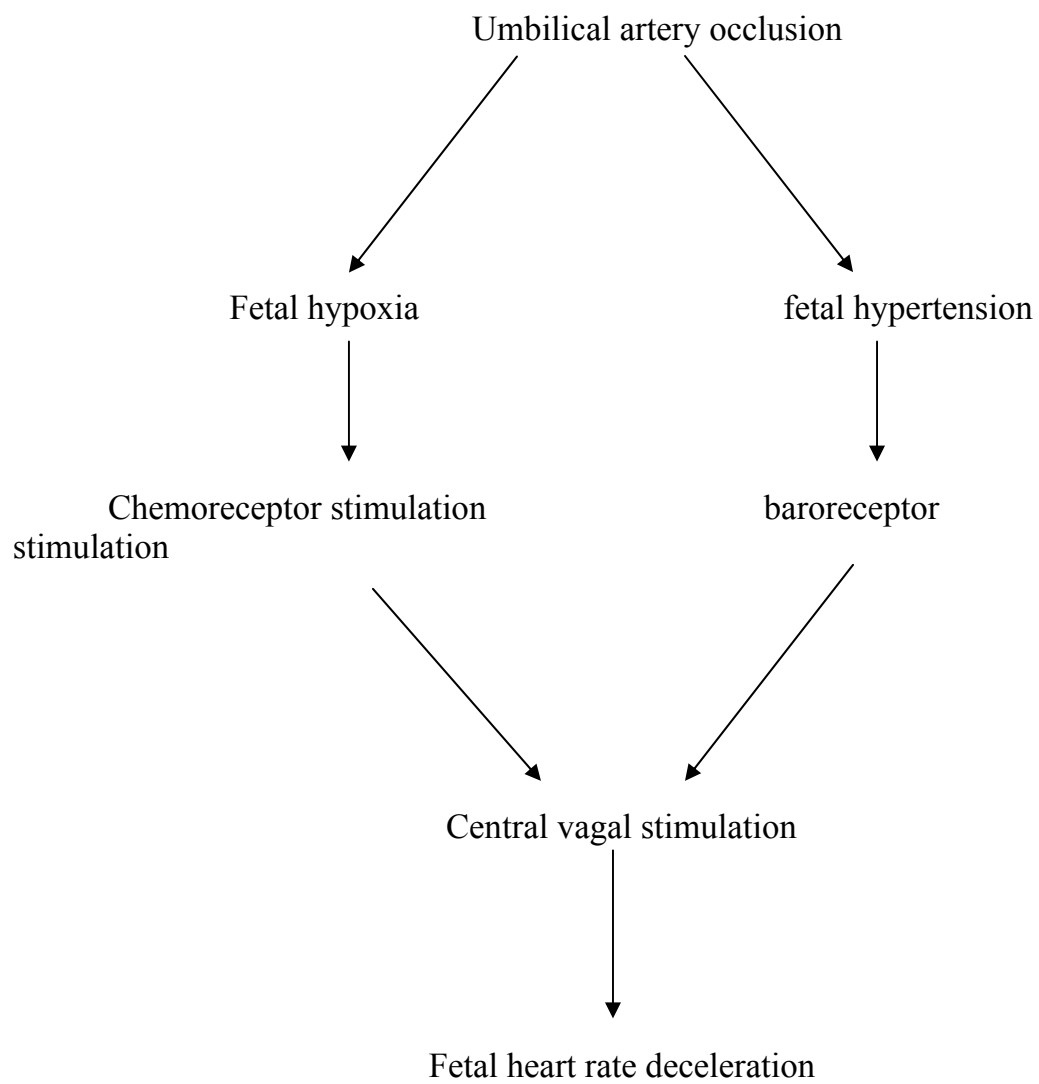
b) Chemoreceptor mediated vagal reflex

Causes:

- 1) fetal hypoxia (uteroplacental induced)
- 2) maternal hypotension
- 3) placental dysfunction
- 4) variable deceleration
- 5) Secondary to cord compression. ACOG (1995) has defined significant variable deceleration as those decreasing to less than 70 beats per min. and lasting more than 60 seconds.

SHOULDERING: this is characterized by ‘shoulders’ of acceleration before and after the deceleration component. It is due to differing degrees of partial cord occlusion. The occlusion of only the vein reduces fetal blood returns thereby triggering baroreceptor mediated acceleration.

Subsequent complete occlusion results in fetal systemic hypertension due to obstruction of umbilical artery flow. This stimulates a baroreceptor mediated deceleration. Presumably, the after coming shoulder of acceleration represents the same events occurring in reverse.

MECHANISM

FETAL SCALP BLOOD pH ESTIMATION

Fetal scalp blood sampling (FBS) for estimation of pH was introduced by Saling, in 1960. When the fetal heart rate raise is normal, the chance of fetal acidosis is rare (Ingmarsson, 1981) while abnormal fetal heart changes are not associated with acidosis. Hence in all cases of suspicious fetal heart rate trace in labour, FBS to detect acidosis (pH less than 7.2) is recommended.

Even in the presence of ominous fetal heart rate pattern of tachycardia, reduced baseline variability and deceleration, only 50 – 60% of fetus are acidotic.

A single record of normal scalp pH does not guarantee fetal health. Significant hypoxia will produce a progressive acidemia that will be recognized by repeated measurements. In the presence of continuous abnormal fetal heart rate trace FBS should be repeated in 30 min. A downward trend suggests deteriorating fetal oxygenation and requires further evaluation while an abnormal value indicates delivery.

During labour, fetal acidosis may result from impaired fetomaternal exchange. Of great concern is inadequate fetal oxygenation because of impaired respiratory gas exchange in the intervillous space. When there is inadequate fetal oxygenation for energy production and the aerobic processes generating ATP fail, anaerobic pathway of energy production is used. Lactic

acidosis results and fetal pH falls. If sufficient hypoxia and acidosis develop, brain damage or death from asphyxia may occur.

The collection of fetal blood for pH estimation, when performed at the appropriate time, may alert the obstetrician to impending fetal jeopardy and permit correction of underlying problem. Because of the intermittent nature of this technique, many fetal blood samples are needed to assess fetal condition. FBS has a high negative predictive value but only a modest positive predictive value. False positive low pH less than 7.2 arises in the following situation:

- 1) uterine hypertonus
- 2) maternal ketoses
- 3) maternal hypotension

Contraindication

In certain situations, performance of FBS may lead to undue wastage of time.

1. Abnormal fetal heart rate pattern in early labour together with thick Meconium stained amniotic fluid.
2. when the progress of labour is unsatisfactory

3. Prolonged Bradycardia < 80 beats for minute for more than 10 min.
4. Sinusoidal pattern without acceleration.

The pH and base deficit are the most useful values to assess fetal condition.

KREBS INTRAPARTUM SCORING SYSTEM

There are several scoring systems for the assessment of the CST during labour. One such intrapartum scoring system was described by Krebs et al , 1979. Like the APGAR score, there are 5 variables, each given between 0 and 2 points. There is a good correlation between these scores and APGAR scores at birth. Scores between 8 and 10 would be incompatible with fetal asphyxia. Scores between 6 and 7 may indicate a compensated fetal distress. Scores between 0 and 5 are considered abnormal, where the possibility of fetal distress is significant.

This type of scoring system has been criticized for being too complicated for daily clinical practice. An experienced interpreter might not need a scoring system for routine use, but for an untrained observer this helps to distinguish pathophysiological from physiological changes.

The Krebs score has been applied to the analysis of the time taken to develop acidosis (pH less than 7.26) when pronounced variable or late deceleration are present with reduced variability. The records were observed for between 30 and 240 minutes until delivery. Tracings with the score 7 or less indicated assessment by scalp blood pH. During the first interval of 90 to 100 minutes the occurrence of fetal acidosis was rather infrequent, but thereafter increased rapidly. Half of all cases had reached a pH value below 7.26 by 115 minutes, when late decelerations were present; the corresponding time for variable deceleration was 145 minutes and for absent variability was 185 minutes.

INTRAPARTUM CST SCORING SYSTEM (Krebs et al. 1979)

Points	0	1	2
Baseline rate (bpm)	< 100 > 180	100 – 119 161 – 180	120 - 160
Baseline variability			
Oscillatory amplitude (bpm)	< 3	3 – 5 > 25	6 – 25
Oscillatory frequency / min.	< 3	3 – 6	> 6
Accelerations / 30 min.	0	Periodic 1 – 4 sporadic	> 5
Decelerations	Late , severe, variable	Mild or moderate Variable Dip 0	None Early

AIM AND OBJECTIVE

To compare Krebs scoring system for intrapartum fetal surveillance with Scalp blood pH estimation in detecting compromised fetus.

MATERIAL AND METHODS

Study Design : Prospective case control study

Study Period : January 2006 - 2007

This study was conducted in 100 patients at term admitted in the labour ward of Institute of Social Obstetrics and Government Kasturba Gandhi Hospital, Chennai.

This study was approved by hospital ethical committee.

1. **Cardiotocograph :**

The apparatus used in this study is FETAL CARE) cardiotocograph. It consists of

- a) ultrasound transducer
- b) tocodynamometer
- c) transducer belt and buckle set
- d) chart paper roll
- e) USG coupling gel
- f) A.C. line cord

2. For fetal scalp blood pH estimation :

- a) speculum
- b) focusing lamp
- c) sponge holder
- d) 11 blade with BP handle
- e) Perineal sheet
- f) pH paper
- g) Heparinised capillary tube

PROCEDURE

The external form of fetal monitoring consists of ultrasound transducer and tocodynamometer fixed to the maternal abdomen. Patients are placed in left lateral position with a tilt of 30 degrees to prevent supine hypotension. Maximum area of fetal heart sound audibility was chosen and transducer was applied after the application of gel. The recording was done for 20 mins at a speed of 1 mm per minute.

FETAL SCALP BLOOD SAMPLING TECHNIQUE:

Patient is placed in lithotomy position. After draping the perineum speculum introduced. To collect fetal blood chorioamnion must be ruptured and the cervix be sufficiently dilated approximately to 2 -3 cm. The presenting part must be sufficiently low in the pelvis to remain reasonably mobile. When the fetus is presenting as a breech, fetal blood can be obtained from the buttock. The fetal scalp is wiped with cotton swab.

An incision is made through the skin to a depth of 2 mm. with the blade. A drop of blood is collected immediately into a heparinised glass capillary tube and pH is measured with pH meter. After the fetal blood sample has been taken pressure should be applied to the puncture site by cotton swab for two consecutive contractions.

If the pH is greater than 7.25 labour is observed.

If the pH is between 7.20 to 7.25 (PREACIDOSIS) repeat again in 30 minutes. If the pH is less than 7.20 (ACIDOSIS) prepare for emergency delivery.

COMPLICATIONS

1. Scalp haematoma
2. Scalp infection
3. Abscess formation

METHODS

200 patients in term labour admitted in labour ward were selected based on the selection criteria and submitted to Krebs intrapartum scoring. All of them were in active phase of labour. Those with Krebs scoring $<$ or equal to 7 were taken up for scalp pH estimation.

In our study 50 patients who had Krebs score $<$ or equal to 7 and were subjected to scalp pH estimation.

INCLUSION CRITERIA

1. Term gestation
2. Single cephalic presentation
3. No other complications of labor
4. No contraindication for vaginal delivery
5. Clear liquor

RESULTS

The results of this study which was conducted in 200 patients in active phase of labour at the labour ward of Institute of Social Obstetrics, Govt. Kasturba Hospital, Madras Medical College, Chennai were as follows:

1. AGE :

Mean	LCL of Mean	UCL of Mean
24.145	23.7253	24.5646

Group	age	No. of cases	%
1	< 20	20	10
2	21 - 25	130	65
3	26 - 30	44	22
4	> 30	5	3

In our study, majority of patients 65% were in 21 – 25 years age group

2. BOOKED OR UNBOOKED :

GROUP	No. of cases	Percentage
BOOKED	198	99
UNBOOKED	2	1

99% of the patients taken up for study were booked cases. Only 1% of the study population was unbooked

3. **PARITY :**

Parity	Group	No. of cases	%
Primi	1	131	65.50
Second gravida	2	51	25.50
Third gravida	3	16	8
Fourth gravida	4	2	1

Primi gravida constituted the majority of the study population (65%).

4. KREB'S SCORING :

N	Mean	S.D	S.E
200	7.72	2.35	0.16

LCL: 7.39152

UCL: 8.04847

Krebs score	No. of cases	%
≤ 7	50	25
> 7	150	75
total	200	100

Out of 200 cases subjected to the Krebs intrapartum scoring system, 150 had scores above 7 while 50 patients had score less than or equal to 7. 25% of the study population had scores less than or equal to 7 while majority (75%) of the study population had scores greater than 7.

5. SCALP BLOOD pH ESTIMATION :

N	Mean	S.D	S.E
50	7.2032	3.8249	5.4092

LCL: 7.19232

UCL: 7.21407

Group	pH value	No. of cases	%
1	> 7.25	10	20
2	7.2 - 7.25	8	16
3	< 7.2	32	64
TOTAL		50	100

50 patients with abnormal Krebs scores were subjected to scalp blood pH estimation. Out of them 10 cases (20%) had pH value greater than 7.25 while 32 cases (64%) had pH less than 7.20 (acidotic pH), while 8 cases (16%) had pH 7.20 – 7.25. 20 Patients with Krebs scores > 7 were selected and scalp blood pH was done. All of them had pH >7.25.

6. COMPARISON OF KREBS SCORING WITH APGAR :

Krebs score	APGAR < 7	APGAR > 7	TOTAL
≤ 7	31	19	50
> 7	5	145	150
TOTAL	36	164	200

$$\text{Sensitivity} = a / a+c = 31 / 36 = 86.111$$

$$\text{Specificity} = b / b+d = 145 / 164 = 88.414$$

$$\text{Positive predictive value} = a / a+b = 31 / 50 = 62\%$$

$$\text{Negative predictive value} = d / c+ d = 145 / 150 = 96.66\%$$

Cases with Krebs score less than or equal to 7 (50 cases) had APGAR less than 7 in 31 cases and APGAR greater than 7 in 19 cases. Whereas cases with Krebs score greater than 7 have APGAR > 7 in 145 cases and APGAR < 7 in 5 cases. The sensitivity is 86.11%. Positive predictive value of Krebs score is 62% whereas negative predictive value is 96.66%.

7. COMPARISON OF SCALP BLOOD pH ESTIMATION WITH APGAR :

pH value	APGAR < 7	APGAR > 7	TOTAL
< 7.25	29	9	38
> 7.25	7	5	12
TOTAL	36	14	50

$$\text{Sensitivity} = a / a+c = 29 / 36 = 77.77\%$$

$$\text{Specificity} = d / b+d = 5 / 14 = 35.7142 \%$$

$$\text{Positive predictive value} = a / a+b = 29 / 38 = 76.31 \%$$

$$\text{Negative predictive value} = d / c+d = 5 / 12 = 41.66\%$$

Out of 50 cases subjected to scalp blood pH estimation, cases with pH less than 7.25 had APGAR less than 7 in 29 cases, while APGAR greater than 7 in 9 cases. The sensitivity is 77.77%. The positive predictive value is 76.31% while negative predictive value is 41.66%.

8. COMPARISON OF KREBS SCORE WITH MODE OF DELIVERY :

Krebs score	Group	IVD		LSCS		Normal		Total
		No	%	No	%	No	%	
> 7	1	4	2.66	16	10.66	130	86.66	150
≤ 7	2	5	10	25	50	20	40	50

IVD – Instrumental Vaginal Delivery

P value = 0.0000 (Using Chi-square test) Significant.

Out of 50 cases with Krebs score <7 normal delivery constitutes 40%, LSCS 50% and Forceps 10%. Whereas when Krebs score >7 normal delivery constitutes 86.66% , LSCS 10% and Forceps delivery 2.66%. There is a high chance of normal delivery when score >7.

9. COMPARISON OF SCALP BLOOD pH ESTIMATION WITH MODE OF DELIVERY :

Scalp blood pH	IVD		LSCS		Normal		TOTAL
	No	%	No	%	No	%	
> 7.25	0	0	0	0	10	100	10
< 7.25	5	12.5	25	62.5	10	25	40
TOTAL	5		25		20		50

N = 50

Out of 50 patients subjected to scalp pH estimation, those with pH >7.25 had 100% normal delivery (10 cases). When pH value <7.25, there is considerable increase in LSCS (62%). Normal delivery constitutes 25% whereas forceps constitutes 12.5%. There were 8 cases with a pH value of 7.20 – 7.25. Out of which 4 delivered within half an hour, while for the remaining 4 patients pH value was repeated after 30 min. Two of them had pH value in the acidotic range and hence taken up for LSCS. Remaining 2 of them had same pH value (same as first value) and delivered by forceps.

**10. COMPARISON OF KREBS SCORING AND FETAL SCALP pH
WITH MODE OF DELIVERY:**

	IVD		LSCS		Normal		Total
	N	%	N	%	N	%	
Krebs	9	4.5	41	20.5	150	75	200
Scalp pH	5	10	25	50	20	40	50
Total	14		66		170		

P = 0.0000 (Chi-square test) SIGNIFICANT.

This table relates Krebs's score with scalp pH significantly in terms of mode of delivery. Using Krebs score there is significant reduction in caesarean rate (20.5%) compared with scalp pH estimation (50%). There is an increase in normal delivery in which the Krebs score is more than 7 (75%) compared to scalp blood pH (40%).

**11.COMPARISON OF KREBS SCORING WITH NICU
ADMISSION:**

Krebs score	Admitted		Not admitted		Total N
	N	%	N	%	
> 7	0	0	150	100	150
< 7	36	72	14	28	50
Total	36		164		200

Krebs score> 7 has no NICU admission whereas Krebs score less than 7 has 72 % admission in NICU.

**12.COMPARISON OF SCALP BLOOD pH WITH NICU
ADMISSION :**

pH	Admitted		Not admitted		Total
	N	%	N	%	
> 7.25	0	0	10	100	10
< 7.25	36	90	4	10	40
Total	36		14		50

Scalp blood pH > 7.25 had no NICU admission, while pH less than 7.25 had 90% NICU admission.

13.COMPARISON OF KREBS SCORE WITH NEONATAL COMPLICATIONS :

Krebs score	Birth asphyxia 1		Respiratory distress 2		Others 3		Nil complications 4		Total
	N	%	N	%	N	%	N	%	
> 7	0	0	0	0	2	1.33	148	98.66	150
< 7	32	64	6	12	2	4	10	20	50
Total	32		6		4		158		200

Cases with Krebs score > 7, congenital anomalies were noted in 2 babies. Remaining 148 had no complications. In cases with Krebs score < 7, 32 babies were admitted for birth asphyxia, 6 had respiratory distress and one admitted for hypoglycemia and the remaining one admitted for congenital anomalies.

**14.COMPARISON OF SCALP BLOOD pH WITH NEONATAL
COMPLICATIONS :**

pH value	Birth asphyxia 1		Respiratory distress 2		Others 3		Nil complications 4		Total
	N	%	N	%	N	%	N	%	
> 7.25	0	0	2	20	2	20	6	60	10
< 7.25	32	80	6	15	2	5	0	0	40
Total	32		8		4		6		50

In cases with pH > 7.25, two had respiratory distress, two had congenital anomalies and 6 did not have any complications. Whereas in cases with pH less than 7.25, birth asphyxia occurred in 32 cases, respiratory distress in 6 cases, one had congenital anomaly and one went in for hypoglycemia.

**15.COMPARISON OF SCALP BLOOD pH ESTIMATION WITH
FETAL OUTCOME :**

pH	Alive 1		Dead 2		Total
	N	%	N	%	
> 7.25	10	100	0	0	10
< 7.25	34	85	6	15	40
Total	44		6		50

This table compares the scalp blood pH estimation with fetal outcome. Out of 50 cases done, 10 had pH values greater than 7.25 and fetal outcome was good (100%). In remaining 40 patients with pH less than 7.25, 6 (i.e. 15%) neonatal deaths occurred.

16. COMPARISON OF KREBS SCORE WITH FETAL OUTCOME:

Krebs score	Alive 1		Dead 2		Total
	N	%	N	%	
> 7	150	100	0	0	150
< 7	44	88	6	12	50
Total	194		6		200

Patients with Krebs score > 7, had 100% good fetal outcome whereas those with score less than 7 had 6 neonatal deaths.

17. BIRTH WEIGHT:

BIRTH WEIGHT	No. of cases	%
< 2.5 Kg.	20	10
2.5 – 3.0 Kg.	150	75
> 3 Kg.	30	15
Total	200	100

Majority of neonates belong to the group of 2.5 to 3.0 Kg.

DISCUSSION

This study was conducted at Institute of social Obstetrics, Govt. Kasturba Gandhi Hospital, in 200 term patients, admitted in labour ward to assess the validity of the Krebs scoring in intrapartum fetal surveillance compared with scalp blood pH, during the period of Jan, 2006 to Jan, 2007. All of them were in active phase of labour.

Among 200 patients, Krebs intrapartum scoring with CST was done. 150 patients had reactive score > 7 , while 50 patients had scores ≤ 7 . Scalp blood pH was taken for these 50 patients.

Table 1,2 AND 3: In this study 65% were in 21-25 years age group and majority were primi gravida (65%).

99% of the study population was booked.

Table 4 AND 5: Kuble and colleagues (1969), studied that in negative CST, pH more than 7.25 was seen in 80% of cases. In our study, out of 50 cases with negative CST values, 10 patients had pH > 7.25 (20%).

In a study by Westgren et al 1980, 20% had abnormal CST in low risk population. In this study out of 200 cases ,50 patients had abnormal CST(25%).

In a study by Beard et al. (1971), Tejani et al. (1975), acidosis was present in 50% of cases with negative CST.

According to P T Steer et al. (1989), the sensitivity of an abnormal CST at any time for acidosis was 80%.

In this study out of 50 patients with negative CST pH less than 7.2 is seen in 32 cases. pH 7.2-7.25 is seen in 8 cases.

Hence pH <7.25 is seen in 80% of cases.

Table 6 and 7 : According to ACOG in 1999, a normal CST has a 99.7 % predictive value of an APGAR score > 7, whereas abnormal fetal heart rate pattern confers a 50 % negative predictive value of Apgar < 7.

EFM has high sensitivity but it's specificity is low (Mongelli et al , 1997). A Normal CST pattern carries a predictive value of over 95% for APGAR > 7 while an abnormal pattern carries predictive value of 60% for APGAR < 7 (Banta and Thakur,1979).

In this study the positive predictive value of Krebs score was 62%, and negative predictive value is 96.66%.

In a admission test conducted at Kandang karabu Hospital, in low risk cases, APGAR less than 7 was present in 1.4% in cases with positive CST. In patients with negative CST, APGAR < 7 was present in 50% of cases. In our

study, in patients with reactive CST, APGAR < 7 was present in 5%. In negative CST, APGAR score of <7 was present in 62% of cases.

According to Arulkumaran and Gibbs (1990), APGAR score of < 7 was present in 40 % cases in negative CST. In positive CST cases, APGAR < 7 was seen in 1.4%.

In 1982, Skyes et al reported that 27% of babies with severe acidosis had APGAR <7. In our study out of 50 cases with pH less than 7.25, 29 babies had APGAR less than 7 (62%).

Similar study was conducted by Nagil et al and reported 30 newborn with pH < 7. All but 10 had APGAR seven or more.

In this study 38 patients had pH < 7 out of them 9 patients had APGAR > 7 while 29 had APGAR < 7. Hence all patients with pH < 7 did not have a low APGAR score (i.e. < 7).

Skyes et al in a study from Oxford showed that if the end point is taken as APGAR < 7 , fetal blood sampling has high negative predictive value and only modest positive predictive value. In our study, sensitivity of fetal scalp blood pH sampling is 77.77%. Positive predictive value is 76.31% negative predictive value is 41.66%.

Table 8, 9 and 10 : Thacker et al (1979) studied that there is a reduced rate of caesarean section in reactive CST pattern. In our study out of 150 patients with Krebs score >7 , 16 patients had caesarian section (10.6%), whereas 50% had caesarean section in Krebs score <7 .

In a study conducted by Tejani et al, addition of fetal blood sampling reduces caesarean sections from 10.05% to 4.5%. In our study, the addition of fetal blood sampling did not have any effect on the rate of CS.

Murphy et al 1990, in his study has shown the caesarian section rate of 10% in cases with reactive CST. In this study, out of 150 patients with reactive CST, 10% had section .86% had normal delivery and 2.6% had IVD.

Continuous EFM has contributed to an increased rate of caesarean section (Clark et al 1982). But a study by Arul Kumaran has shown unchanged caesarean section rate for fetal distress (1-2%) using intrapartum CST. In our study caesarean section rate using CST is 20.5%.

Out of 5000 low risk population delivered at University hospital, Lund, Sweden during 1977 -1978, only 30 patients (0.65%) were delivered by caesarean section for fetal distress using CST.

In our study out of 200 patients monitored by CST only 25 had LSCS for fetal distress.

Zuspan et al 1979, showed that FBS should be used in conjunction with EFM, to improve specificity. When FBS is not used to verify the fetal acid base status there is an increase in caesarean section for presumed fetal distress. (Grant et al 1989)

In our study out of 50 patients with abnormal CST 25 had LSCS. The remaining 5 had instrumental vaginal delivery and 20 had normal delivery.

Table 11 and 12 : According to National Institute of Health and Human development Research, planning workshop report, 1999, a normal fetal heart rate tracing conferred a high predictability for a well oxygenated fetus. In their study neonatal admission in normal CST group is 6%, in abnormal CST group is 38%. In this study there were no NICU admission in normal CST patients and it was 72% when the CST was abnormal.

In a study by Skyes et al neonatal admission was noted in 14.3% of acidotic pH cases and 1.4% of normal pH cases. When pH was <7 , we had 90% neonatal admissions.

Table 13 and 14 : Most records with ominous fetal heart changes were not associated with fetal acidosis in labour (Arul kumaran and Ingermasson). Low scalp blood pH may be found in 50% of cases with ominous CST changes, but in a considerable proportion of these cases, the fetus has a mild or a moderate respiratory acidosis which is not associated with asphyxia (40% , Ingermasson).

Vandenberg et al reviewed the neonatal complications of 84 babies with pH less than 7 with non acidotic pH babies (>7). They found highly significant differences in babies with as to birth asphyxia between epidotic (80%) and non epidotic babies.

In this study out of 40 cases with pH <7.25 , 32 had birth asphyxia (80%), 15 had respiratory distress and 5% had other complications.

Winkler et al 1989 found out that only 23 term babies with a pH < 7 ,had significant birth asphyxia.

In our study out of 50 babies with pH <7.25 40 babies had birth asphyxia (80%).

Goldabar et al 1991, reported that 2 out of 3 term newborn had an uneventful neonatal period despite pH < 7.25 . The fetal outcome was significantly better with pH value >7 . In this study, out of ten patients with pH >7 only 2 patients had respiratory distress.

In our study the neonatal outcome in all the 150 patients who had normal CST (Krebs score > 7 was good (96.66%). Hypoxia and acidosis are unlikely in the presence of normal reactive fetal heart rate pattern (Ingermasson et al 1993) especially when labour is progressing satisfactorily.

In our study, rate of fetal distress in low Krebs score group of patients was 12% (6 out of 50 patients), whereas in patients with scalp blood pH $<$

7.25, the rate is 15% (6 out of 40 patients). This shows that Scalp blood sampling for pH estimation only marginally increased the rate of detection of fetal distress.

Table 15 and 16 : Arul kumaran and Gibb (1990) reported that intrapartum hypoxia was responsible for 14.5% of perinatal deaths in 1982, when intermittent auscultation was used. After introduction of continuous electronic fetal monitoring perinatal deaths came down to 7.9%. Edington et al 1975, Biswas et al showed similar reduction in perinatal mortality. In our study only 6 babies died.

Zuspan et al 1979 studied that reactive CST is associated with good outcome (98%). In our study reactive CST (Krebs score >7) is associated with 100% good fetal outcome.

Westgren et al (1980) showed that there was no fetal death in a low risk population of 5037 women who had reactive CST. In our study, out of 150 patients with Krebs score >7, there was no fetal death.

Goldaber et al 1991, has shown 8% fetal death in his study with pH <7. In this study, Death rate is 12%.

CONCLUSION

This study on validation of Krebs scoring system for intrapartum CST in detecting compromised fetus compared to scalp blood pH estimation, shows the superiority of Krebs's scoring for early detection of fetal jeopardy when compared to fetal blood sampling.

Fetal blood sampling has its own limitations - mainly –restricted availability, need for rupture of membranes (Usually late in labor) and the time lag between sampling and result

Krebs intrapartum scoring system is also not without its share of disadvantages in that it needs expertise in interpreting the CST and very often, a positive CST might be considered negative by inexperienced persons.

However when Krebs scoring system and FBS are combined together, the outcome is much better and there is a definite reduction in fetal compromise.

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PROFORMA

Date :

Name :

Age :

IP no. :

Parity:

Booked or unbooked

LMP:

EDD:

Gestational age:

Any associated medical complications:

Clinical examination:

Height:

Weight:

Fundal height:

Fetal heart rate:

Presentation:

Colour of liquor:

Type of labour: spontaneous / induced

Bishop's score:

Duration of labour: I stage

II stage

Krebs score: group 1 (>7)

Group 2 (≤ 7)

Scalp blood pH: group 1 (pH above 7.25)

Group 2 (pH 7.20 – 7.25)

Group 3 (pH < 7.20)

Repeat pH:

Time of delivery:

Mode of delivery: F – Instrumental vaginal delivery

L – LSCS

N – Normal delivery

Birth weight : group 1 (< 2.5 Kg.)

Group 2 (2.5 – 3.0)

Group 3 (> 3)

APGAR group : group 1 (>7)

Group 2 (<7)

Associated neonatal complications: group

1. birth asphyxia
2. respiratory distress
3. Others (e.g. Congenital anomalies)

NICU admission: yes / no

Fetal outcome: 1.alive 2.dead